

*The Riskscape and the Color Line:
Examining the Role of Segregation
in Environmental Health Disparities*

Rachel Morello-Frosch
Brown University
Center for Environmental Studies &
Department of Community Health, School of Medicine

&

Russ Lopez
Boston University
Department of Environmental Health, School of Public Health

- Prepared for the U.S. Environmental Protection Agency, Office of Children's Health Protection (Contract No. EP-W-04-049, Task Order No. 101), in preparation for the Workshop: *Methods for Assessing Environmental Health Disparities*, May 24-25 2005, Ann Arbor, MI.

Draft – Please do not cite or circulate without permission from the authors

May 2005

Table of Contents

| | |
|--|----|
| Executive Summary | i |
| Introduction..... | 1 |
| Environmental Health Disparities in the Context of Neighborhoods and Regions | 3 |
| A Conceptual Framework for Linking Segregation to Environmental Health Disparities. | 7 |
| The Dimensions and Measurement of Racial Residential Segregation | 10 |
| Analytical Applications of Segregation Measures in Environmental Health | 17 |
| Applying Segregation Measures to Study Other Environmental Hazards and Health Outcomes | 23 |
| <i>Lead & Residential Pesticide Use</i> | 24 |
| <i>Stationary and Mobile Pollution Sources</i> | 25 |
| Segregation in Relation to Health Outcomes That May be Environmentally Mediated .. | 26 |
| <i>Adult Mortality</i> | 27 |
| <i>Infant Mortality and Other Birth Outcomes</i> | 28 |
| <i>Asthma</i> | 30 |
| Conclusions and Implications for Policy | 31 |
| Figures and Tables | 36 |
| References..... | 46 |

Executive Summary

Environmental health researchers, sociologists, policy-makers, and activists concerned about environmental justice argue that communities of color who are segregated in neighborhoods with high levels of poverty and material deprivation are also disproportionately exposed to physical environments that adversely affect their health and well-being. Examining these issues through the lens of racial residential segregation can offer new insights into the junctures of the political economy of social inequality with discrimination, environmental degradation, and health. More importantly, this line of inquiry may highlight whether observed pollution – health outcome relationships are mediated by segregation and whether segregation patterns impact diverse communities differently.

Although elements for understanding the relationship between residential segregation and community environmental health can be found separately in both the sociology literature and the environmental justice literature, only one previous investigation has attempted to combine these two lines of inquiry to analyze the relationship between outdoor air pollution exposure and segregation (Lopez 2002). Some researchers have recently argued that residential segregation is a crucial place to start for understanding the origins and persistence of environmental health disparities. This paper, commissioned for a workshop on developing measures to research and track environmental health disparities, examines theoretical and methodological questions related to racial residential segregation and environmental health. We seek to address the following questions: 1) Which metrics for measuring segregation are appropriate for

the study of environmental health disparities? Are the metrics universally applicable across the range of environmental health issues and ethnic groups? 2) Can the methods applied to assess the relationship between segregation and air pollution be used for other exposures and health issues? 3) Given that most measures of segregation consider only dyads, to what extent are existing measures of segregation valid for multi-ethnic regions?

There are five primary dimensions of segregation, (evenness, isolation/exposure, clustering, concentration and centralization), all of which have varying conceptual implications for environmental health research and assessing disparities in exposures and health outcomes that may be environmentally mediated. Evenness is the measure that has been most frequently used in the sociology and public health literature and applied to various contexts (e.g. schools, the workplace, and neighborhoods). This measure estimates the degree to which the proportion of a particular racial or ethnic group living in residential areas (e.g. census tracts) approximates that group's relative percentage of an entire metropolitan area. The isolation or exposure indices are perhaps the best measure for reflecting how members of racial groups actually experience residential segregation in their neighborhoods but, unlike evenness, these measures are composition dependent. (Farley 1984) The other three dimensions of residential segregation, which tend to characterize the spatial dimensions of segregation within metro areas (such as concentration, centralization, clustering) have been used less frequently. However these measures may be particularly useful when examining environmental health questions, particularly when focusing on a small number of MSAs that may be similar compositionally and in overall size. These latter measures may help researchers better

grasp how the spatial form of segregation may disproportionately expose certain groups to specific environmental stressors that ultimately degrade community health.

Nearly all of the segregation measures focus on dyadic racial/ethnic comparisons with Non-Hispanic Whites generally serving as the referent group. However, generalized measures can also be used to assess patterns of segregation in a context of diversity where multiple racial/ethnic groups are simultaneously segregated from one another. Here we demonstrate how dyadic and generalized measures of segregation can be applied to examine racial disparities in air pollution burdens in major metropolitan areas of the United States. Based on these results we suggest ways in which segregation measures can be applied to track and research disparities related to other environmental hazards and health outcomes, such as childhood lead exposure and urban pesticide use, the location of mobile and stationary pollution sources, infant mortality and other birth outcomes, and asthma. We also propose a conceptual framework for understanding how segregation may shape the distribution of environmental health disparities and enhance the vulnerability of segregated communities to the adverse health effects of hazardous physical and social environments. We suggest that a regional equity perspective helps elucidate how segregation patterns can create and amplify environmental health disparities. The rationale for taking a regional perspective are twofold: First, previous research strongly suggests that it is more fruitful to assess potential drivers of environmental health disparities at the regional level because economic trends, transportation planning, and industrial clusters tend to be regional in nature, and zoning, siting, and urban planning decisions tend to be local. Second, research that examines

how health inequities play out regionally could lead to interventions and policy initiatives that better bridge the divide between the city core and suburbs and more effectively ameliorate fundamental drivers of environmental health and disease among diverse communities.

Questions to Consider:

- 1) What are the various contexts researchers should consider when examining relationships between segregation and environmental health disparities?
- 2) What are the implications of segregation in the context of different individual risk factors for health outcomes that may be socially and environmentally mediated?
- 3) Should we consider segregation as a potential mediator between environmental hazard exposure and health outcome relationships, or should it be conceptualized as a risk factor in and of itself?
- 4) How should we track and measure segregation in relationship to other key variables that measure socioeconomic status?

Introduction

“The color line is not static; it bends and buckles and sometimes breaks.”

(Drake and Cayton, 1945: 101).

Race, as a social construct and mechanism of classification, has historically defined and continues to shape the distribution of power, privilege, and economic resources in American society (Lawrence 1987; Crenshaw 1988; Wellman 1993; Jones 2001). Myriad forms of discrimination in the U.S. are imprinted onto our urban landscape, as evidenced by the persistent spatial separation of diverse communities along racial/ethnic and, to a lesser extent, class lines (Walker 1981; Logan and Molotch 1987; Massey and Denton 1993; Massey and Gross 1994; Farley 1995; Jargowsky 1997). Wide-ranging and complex political, socioeconomic and discriminatory forces coupled with patterns of industrialization and development have segregated people of color, particularly African Americans, into neighborhoods with some of the highest indices of urban poverty and deprivation (Peet 1984; Walker 1985; Schultz, Williams et al. 2002). Indeed, uneven industrial development, real estate speculation, discrimination in government and private financing, as well as exclusionary zoning have led to systemic racial segregation among diverse communities with important implications for community health and individual well-being (Logan and Molotch 1987; Harvey 1989; Wilson 1996; Sinton 1997; Bobo 2001; Morello-Frosch 2002b; Massey 2004). The socioeconomic effects of urban segregation are further amplified by racialized boundaries in the workplace and in some regions through policies such as immigration law and welfare reform (Hersh 1995; Pulido, Sidawi et al. 1996; Morello-Frosch 2002b).

Although elements for understanding the relationship between residential segregation and community environmental health can be found separately in both the sociology literature and the environmental justice literature, only one previous investigation has attempted to combine these two lines of inquiry to analyze the relationship between outdoor air pollution exposure and segregation (Lopez 2002). Some researchers have recently argued that residential segregation is a crucial place to start for understanding the origins and persistence of environmental health disparities (Morello-Frosch, Pastor et al. 2001; Lopez 2002; Morello-Frosch RA 2002; Morello-Frosch 2002b; Gee and Payne-Sturges 2004). This paper, commissioned for a workshop on developing measures to research and track environmental health disparities, examines theoretical and methodological questions related to racial residential segregation and environmental health. We seek to address the following questions: 1) Which metrics for measuring segregation are appropriate for the study of environmental health disparities? Are the metrics universally applicable across the range of environmental health issues and ethnic groups? 2) Can the methods applied to assess the relationship between segregation and air pollution be used for other exposures and health issues? 3) Given that most measures of segregation consider only dyads, to what extent are existing measures of segregation valid for multi-ethnic regions? To address these questions, the paper first provides an overview of race-based segregation in the United States and proposes a framework for understanding its implications for environmental health disparities. The following section summarizes the various dimensions of residential segregation and the relative limitations and advantages of each for examining environmental health issues. We then

discuss applications of segregation measures for assessing disparities in ambient air pollution burdens across racial groups and go on to discuss the applicability of these methods for other environmental exposures and health outcomes. Finally, we conclude with a short discussion of the policy and regulatory implications of integrating measures of residential segregation to research and track fundamental drivers of environmental health disparities.

Environmental Health Disparities in the Context of Neighborhoods and Regions

The burgeoning literature on health disparities has compelled researchers to move beyond proximate causes of poor health toward identifying fundamental socioeconomic drivers of health and disease in populations (Link and Phelan 1995; Kaplan and Lynch 1999; House and Williams 2000; Navarro 2002). This requires examining how the socioeconomic conditions of residential environments affect health and well-being. Indeed, research strongly suggests that place matters to health (Yen and Syme 1999; Macintyre, Ellaway et al. 2002). Yet, despite the proliferation of work on the issue of segregation, there is a lack of scientific consensus about what it is about neighborhoods, and segregated neighborhoods in particular, that affects health. Neighborhood-level factors associated with racial residential segregation may affect individual health by influencing individual food security (access to quality foods, especially fresh fruits, vegetables, and affordable markets) access to crucial services (such as health care, parks, and open space) (Diez-Roux 1997; Center for Third World Organizing 2002; Morland, Wing et al. 2002) the social environment (social capital, cohesion, crime rates, and abandoned properties) (Sampson 1987; Wallace 1990; Conley 1999; Keister 2000;

Kawachi and Berkman 2003), and the physical environment (traffic density and housing quality) (Reynolds, Von Behren et al. 2002; Shenassa, Stubbendick et al. 2004).

Environmental health researchers, sociologists, policy-makers, and activists concerned about environmental justice argue that communities of color who are segregated in neighborhoods with high levels of poverty and material deprivation are also disproportionately exposed to physical environments that adversely affect their health and well-being. Examining these issues through the lens of racial residential segregation can potentially offer new insights into the junctures of the political economy of social inequality with discrimination, environmental degradation, and health. Assessing the various dimensions of segregation within and between metropolitan areas can offer insights into how diverse legacies of discrimination may shape current spatial distributions of pollution sources among diverse communities. More importantly, this line of inquiry may highlight whether observed pollution – health outcome relationships are mediated by segregation and whether segregation patterns affect diverse communities differently. These are all issues worthy of consideration when examining disparities in certain types of environmental hazard exposures, whether the issue is proximity to certain hazardous facilities, isolation and concentration in areas with a high proportion of substandard housing, or neighborhoods where facilities expose local communities to certain pollutants with a propensity to create localized “hotspots.”

Focusing on segregation also promotes a regional perspective for understanding the dynamics that create environmental health disparities. For example, conventional

theories regarding regional development suggest that the formation of large cities in the United States was consonant with a long process in the past of industrial agglomeration in the urban core followed by a more recent countervailing trend of industrial suburbanization. The morphology of the urban landscape is also shaped by shifting patterns of capital and state investment; government at the local, state, and federal levels often promotes industrial expansion by facilitating investment flows to outlying regional areas through highway construction and other infrastructure projects, tax breaks, and mortgage subsidies (Logan and Molotch 1987; Hise 1997). Historically, working-class and poor communities of color have been spatially bound in this process, remaining close to large production facilities, because of limits imposed by job search, work hours, income, and exclusionary and discriminatory housing development policies (Massey and Denton 1993; Guhathakurta and Wichert 1998).

Imposed limitations on the spatial mobility of certain groups also directly undercuts their economic mobility because of the close connection between these two phenomena (Massey and Fong 1990; Massey, Gross et al. 1991). Indeed, the historical and contemporary racial segmentation of the housing market has eroded the property values of Black housing and limited the capacity of Black families to accumulate wealth through home equity (Oliver and Shapiro 1995; Conley 1999). Segregation can also cause so-called “spatial mismatch” between the location of lucrative jobs and the residential location of the communities that need them (Kain 1968; Preston and McLafferty 1999). Conversely, wealthier, mostly White, classes enjoy the mobility and privilege to pursue emerging economic opportunities and to escape the toxic zones of

industrial activity (Pulido 2000). Therefore, segregation can play out in ways in which certain groups become concentrated, centralized, and isolated into abandoned inner city cores where employment opportunities are few and where communities are clustered around industrial sites or transportation corridors that pose significant health hazards.

The historical processes that create racialized territories within metropolitan areas are further amplified by current policies of uneven regional industrial development, which leads to patterns of environmental discrimination. For example, in the Los Angeles region, Pulido and colleagues argue that development in Torrance, led by large petroleum companies and metal industries, was strictly zoned to exclude residential land uses, except for a small area of Mexican housing known as “the Pueblo Lands.” Companies implemented a division of labor along racial lines where skilled jobs were the purview of White workers and low-skilled, low-wage, and more hazardous jobs were filled by Latino laborers. Occupational segregation coupled with discriminatory zoning strategies and real estate covenants, assured the physical separation of Latino and White residents (Pulido, Sidawi et al. 1996).

The structural forces that create segregation tend to operate regionally, as evidenced by many current political and economic regions that are not producing optimal outcomes for communities of color, the working class, and the poor, both in terms of economic growth and environmental quality (Pastor, Dreier et al. 2000; Pastor 2001). Regional equity refers to the notion that metro areas and cities that are integrated along economic, political, and environmental lines have a more equitable distribution of

resources and tend to collectively fare better on a number of important outcomes. Examples of such benefits include a stronger, more stable tax base, healthy communities, and planned land use development (Pastor, Dreier et al. 2000; Pastor 2001). The importance of regional equity can be extended to address regional disparities in health and the potential for improving outcomes by linking together the future of suburbs and cities. Examining the drivers and impacts of economic and racial segregation has become an integral piece of addressing regional equity. From a public health perspective, the rationale for taking a regional perspective when examining links between segregation, environments, and health disparities is twofold: First, previous research strongly suggests that it is more fruitful to assess potential drivers of environmental health disparities at the regional level because economic trends, transportation planning, and industrial clusters tend to be regional in nature, and zoning, siting, and urban planning decisions tend to be local (Morello-Frosch, Pastor et al. 2002a). Second, research that examines how health inequities play out regionally could have implications for the development of localized interventions and policy initiatives that ameliorate fundamental drivers of environmental health and disease among diverse communities.

A Conceptual Framework for Linking Segregation to Environmental Health Disparities

We propose a framework for understanding the relationship between racial residential segregation and various indicators of environmental health inequalities. Building on frameworks proposed by other health inequality researchers (Schultz, Williams et al. 2002; Gee and Payne-Sturges 2004), Figure 1 conceptually demonstrates

how structural mechanisms leading to residential segregation can shape patterns of differential exposure to environmental hazards, and amplify disparities for myriad health outcomes and differential ability to recover from hazardous exposures. The figure proposes an ecosocial or biosocial framework (Krieger 1994; Krieger 1999; Massey 2004) in order to connect a persistent form of structural discrimination (i.e. racial segregation) to the community-level conditions that may disproportionately expose communities of color to environmental hazards and stressors that enhance individual-level vulnerability to the toxic effects of pollution. This dynamic partially explains persistent racial and class-based health disparities that may be environmentally mediated.

The top of the figure shows the structural mechanisms leading to race-based residential segregation and the resulting community-level and individual-level factors that can influence disease burdens among diverse populations. Segregation solidifies racial disparities in socioeconomic status (SES), and it shapes the distribution of resources and wealth at the individual, household and community levels with important implications for community health. The bottom of the figure shows how these multi-level factors can influence an exposure-health outcome continuum. This exposure-health outcome continuum connects the emission of a contaminant from a source (e.g., an industrial facility or transportation corridor in a neighborhood) to human exposure via various media (e.g., air or water), body burden and internal dose of contaminants, individual resilience (e.g. through detoxification mechanisms) and the occurrence of a health effect (e.g. an adverse birth outcome, such as low birth weight), and the ability to recover. The framework assumes that environmental contaminants lead to human

exposures that can overcome the body's defense systems and have adverse health effects. This dose, if not effectively metabolized or excreted by the body's detoxifying and/or immune systems, can lead to biological effects that may alter system functioning and damage target organ systems. Individual and community-level stressors shape how these differential exposures play out, including increasing or decreasing absorption, ability to detoxify or recover from toxic exposures, and the ultimate short- and long-term health effects from environmental contaminants. Community- and individual-level stressors and buffers can protect against or enhance vulnerability to the toxic effects of contaminants. These factors can include both social and biological elements, including pre-existing health conditions, socioeconomic circumstances, and psycho-social stress (Brunner 2000; (McEwen and Lasley 2002).

The figure notes examples of stressors that can be measured at the individual and/or community level in conjunction with various measures along the exposure-health outcome continuum. Both of these factors, shaped by segregation patterns, can exert their own unique biological impact, both in terms of amplifying differential vulnerability to the toxic effects of pollutants or by weakening the ability to recover from harmful exposures. Furthermore, the literature suggests that individual- and community-level stressors can potentially amplify contaminant exposure and health outcome relationships (Rios, Poje et al. 1993; Brunner 2000; Gordon 2003; Perera, Rauh et al. 2003; Rauh, Whyatt et al. 2004). Therefore, it is important to examine both levels of stressors (Diez-Roux 1997; Diez-Roux 1998; Diez-Roux 2000) to assess their impact on health outcomes that are both environmentally and socially mediated.

The Dimensions and Measurement of Racial Residential Segregation

There has been substantial attention paid to the development and calculation of segregation measures (Duncan and Duncan 1955a; Duncan and Duncan 1955b). A report by the U.S. Census listed over a dozen measures (Iceland, Weinberg et al. 2002) and the sociological literature on residential segregation in the United States proposes that there are five basic dimensions of racial and ethnic segregation: evenness, isolation, concentration, centralization, and clustering (James and Taeuber 1985; Stearns and Logan 1986; White 1986; Massey and Denton 1988). Table 1 summarizes these measures. Although census tracts and metropolitan statistical areas tend to be the primary macro and micro units of analysis to calculate these measures, segregation measures can also be derived using other units as well (e.g. zip codes or block groups to characterize the segregation of counties). Each dimension outlined below presents a unique, but not mutually exclusive, combination of potential social, economic, and health-related disadvantages.

- *Evenness* measures the degree to which the proportion of a particular racial or ethnic group living in residential areas (e.g. census tracts) approximates that group's relative percentage of an entire metropolitan area (Massey, White et al. 1996). It is measured using the dissimilarity index (D) which is interpreted as the proportion of the racial group of interest that would need to relocate to another census tract in order to achieve an even distribution throughout a metro area. The index measure ranges from 0 (no segregation) to 1 (complete segregation) with

0.6 considered to be highly segregated. This measure is the most extensively used indicator of segregation, both in the sociological literature and in the public health literature (Acevedo-Garcia, Lochner et al. 2003).

- *Isolation/Exposure* assesses the extent to which a member of a particular racial/ethnic group is likely to have contact with members of the same group (isolation) or, conversely, the degree to which different groups would be exposed to each other by sharing common residential areas (exposure) (Massey, White et al. 1996). The point of this measure is to assess the diversity of neighborhoods and capture some assessment of the daily experience of segregation felt by certain racial groups.
- *Concentration* attempts to measure the population density of a certain racial/ethnic group within a metro area. Groups that reside in a small share of the total metro area are considered to be concentrated. This measure tries to encapsulate the notion that minority groups have traditionally been restricted to a small set of residential neighborhoods (Massey and Denton 1988).
- *Centralization* refers to a group's proximity to the center of a metropolitan area, which in some of the nation's older cities is characterized by some of the highest levels of poverty, poor housing quality, and economic abandonment (Massey and Denton 1988).

- *Clustering* characterizes the degree to which census tracts occupied by a particular racial/ethnic group are next to each other (or cluster) in space (Massey and Denton 1988). A high level of clustering implies that minority census tracts are contiguous and form a sort of “ethnic enclave” while a low level of clustering indicates that minority census tracts are fairly spread out throughout a metro area.

The magnitude of measured segregation can be affected by the geographic subunit that is used as the basis for analysis. The smaller the subunit, (e.g. census block groups vs. census tracts vs. zip codes), the higher the segregation scores. This is partially due to how people are grouped into households, then blocks, then block groups, etc.

Households tend to be uni-racial. In geographic information systems and geospatial statistics, the general rule is that closer, smaller units tend to be more alike. In terms of the spatial distribution of racial groups, this implies that people on a given block or area are more likely to be similar to their neighbors. But a part of this scale-measurement issue also reflects the fact that the geographic boundaries between Black-majority, White-majority, or Asian-majority, areas are often very distinct, but these boundaries do not necessarily run along tract or zip code boundaries. It should be noted, however, that the magnitude of these scale effects are small (Iceland and Steinmetz 2003). In general, census tracts have been the primary subunit used to analyze segregation, at least since 1970 when metropolitan area counties began to be parsed out into tracts.

It is important to note that traditional measures of segregation (with the exception of the centralization and clustering indices) are “aspatial” in the sense that neighborhoods

can be arbitrarily re-arranged in physical space without altering the value of the index. Several authors have developed methods that account for the spatial relationships between neighborhoods, as well as the differential distribution of racial/ethnic groups across neighborhoods (Downey 2003; Dawkins 2004; Wong 2004). However, these measures are computationally intensive and the methodology is developing rapidly but with no emerging consensus on which measures are best suited for which purposes. Furthermore, to our knowledge, no studies of “spatial” measures of segregation have yet been applied to understanding the distributions of health outcomes.

Nearly all of the measures described above focus on dyadic racial/ethnic comparisons: Black/White, Asian/White, Hispanic/White and so on. Usually Non-Hispanic Whites serve as the referent group in these comparisons. Although these measures are informative, generalized measures are also needed to gain insights on patterns of segregation in a context of diversity where multiple racial/ethnic groups are simultaneously segregated from one another (Iceland 2004). A way to examine segregation in a more dynamic sense that takes into account the rise of multiracial metropolitan areas is through the generalized dissimilarity index which is a variation of the traditional measure of evenness described above that measures segregation among many racial/ethnic groups simultaneously (Sakoda 1981). Similar to the dyadic similarity index, the generalized dissimilarity index varies from a value of 0, meaning no segregation, to 1, or complete segregation. Additional measures of multigroup segregation indices can be found in several recent reviews (Grannis 2002; Reardon and Firebaugh 2002).

Racial composition, or the existence of census tracts with a high proportion of specific minority groups, has been interpreted as a measure of the magnitude of segregation in a metro area. For example, the percentage of Blacks in a census tract has been used to study the health effects of segregated neighborhoods (Acevedo-Garcia, Lochner et al. 2003) Yankauer 1950; Jackson et al., 2000; Fang et al. 1998). By using racial composition as a way to operationalize segregation, these studies assume that racial composition directly reflects a dimension of racial/ethnic unevenness in a particular metro area. However, racial composition may not always be a true reflection of segregation *per se*. This is because segregation is a contextual measure that depends on the relationship between racial groups in neighborhoods (e.g. census tracts) across a larger geographic area (e.g. a metro area). Thus, while percent minority measures reflect the composition of a particular neighborhood, they do not assess whether a metro area's organization reflects larger dynamics of racial inequality. For example, if a particular neighborhood in City X were composed of over 75% Latinos, this may give the impression that Latinos are highly segregated in that particular city. However, if the entire population of City X is 80% Latino, then the racial composition of that neighborhood merely reflects the larger racial composition of the metro area.

Tables 2 and 3 demonstrate current patterns of segregation among racial/ethnic groups in the United States using 2000 Census data. Table 2 uses a dyadic dissimilarity index, which is the level of inequality in the distribution (or unevenness) of a racial/ethnic group compared to Whites. Given the history of discrimination in the U.S.,

it is not surprising that African Americans experience the highest levels of residential segregation although these have declined slightly over the last twenty years. For other racial/ethnic groups, there has been surprisingly little change in their levels of segregation over the last twenty years.

Table 3 shows current segregation patterns for several racial/ethnic groups simultaneously, through the generalized index of dissimilarity (Sakoda 1981). This measure assesses segregation in the context of diversity, rather than focusing on dyads that compare only one racial group at a time to another group (typically Whites as in Table 2). The generalized index of dissimilarity measures the number of persons of each racial/ethnic group who would need to move into (and out of) their neighborhoods in order to achieve an even racial/ethnic make-up. The table shows the percentage distribution of each racial/ethnic group in metropolitan statistical areas that are extremely segregated, highly segregated or moderately segregated. As can be seen, on a national level, African American residents live in areas with some of the highest levels of multi-group segregation, followed by Whites, Hispanics, and Asians. These disparities vary significantly by geographic region, particularly in the Northeast and the Midwest where segregation levels are highest.

In sum, the choice of which segregation measure to use depends on what dimension is being investigated. In general, segregation measures tend to be correlated, rising and falling in tandem; metropolitan areas measuring high levels of segregation along one dimension also tend to have high scores on the others as well. All of the

measures outlined above have varying conceptual implications for environmental health research and assessing disparities in pollution exposures and outcomes that may be environmentally mediated. The index of dissimilarity has certain advantages, due to its ubiquity and longevity in diverse fields and the fact that it is not composition dependent. This measure can be used to compare a diverse array of metro areas and it is not affected by the relative proportion of the demographic groups being examined. Furthermore, the dissimilarity index is the most common measure used in the public health literature, which makes it more intuitive to interpret both methodologically and conceptually. Using the dissimilarity index also allows quick comparisons to other study outcomes such as educational attainment and housing status. The isolation or the exposure indices are perhaps the best measure for reflecting on the how members of minority groups actually experience residential segregation in their neighborhoods (Farley 1984), but these measures are composition dependent

The other three dimensions of residential segregation, which tend to characterize the spatial dimensions of segregation within metro areas (such as concentration, centralization, clustering) have been used less frequently. However these measures may be particularly useful when examining environmental health questions, particularly when focusing on a small number of MSAs that may be similar compositionally and in overall size. These latter measures may help researchers better grasp how the spatial form of segregation may disproportionately expose certain groups to specific environmental stressors that ultimately degrade community health.

Analytical Applications of Segregation Measures in Environmental Health

Few environmental health issues have been studied in the context of segregation, but air pollution has received some attention. These studies illustrate potential pathways between segregation and environmental health outcomes. They also provide a framework for discussing other potential environmental health problems that have yet to be fully studied in the context of segregation. Below are some examples of the analytical applications where measures of segregation can be used to understand environmental health inequalities, related to outdoor air pollution.

Since the passage of the Clean Air Act over 30 years ago, monitoring of outdoor air quality has become ubiquitous in most metropolitan areas. Most monitoring is focused on the criteria pollutants (carbon monoxide, sulfur dioxide, oxides of nitrogen, particulates, ozone, volatile organic compounds, and lead). The EPA has established concentration limits for these pollutants, and exceedances can bring sanctions and public action to insure compliance. Monitoring is usually limited to a small set of strategically placed locations with the goal of measuring the overall air quality across the entire metropolitan area. Attempts have been made to infer air quality on the local level from regionally placed air monitors, often through interpolating results geographically across census tracts and neighborhoods, but the results have not been strong. This reflects the difficulties of using a half-dozen or so monitoring stations (the reported levels themselves are smoothed so that the results represent annual or monthly averages) to characterize air pollution levels in hundreds of census tracts. This smoothing decreases the variability of air exposures across the studied metropolitan areas and thus severely limit the ability to

discern the effects of segregation. Nevertheless, metropolitan level data allow for studying the association between levels of segregation and overall levels of criteria air pollutants.

In order to examine the relationship between air pollutant levels and segregation for this paper, we undertook a set of preliminary regression analyses using metropolitan area-wide criteria air pollutants levels as dependent variables and segregation and other metro level factors as independent variables. Criteria air pollutant levels for each available metropolitan area were obtained from the U.S. EPA's Aerometric Information and Retrieval System (AIRS) database. The database contains annual metropolitan area-wide averaged levels of selected criteria air pollutants (EPA 2004). Black-White Dissimilarity Index scores were calculated by the Mumford Institute using 2000 Census data (Mumford Center 2000). Other potential metropolitan level explanatory variables, such as the percent of the total population living in poverty, total population, per capita income, percent of civilian labor force employed in manufacturing and percent of Black residents, were obtained from the U.S. Census. Controlling for these SES variables, at the census tract level, Black-White segregation was associated with increased metropolitan-wide levels of sulfur dioxide and ozone. Segregation was also associated with increased levels of PM₁₀, but this association was not significant. Segregation was associated with decreased levels of carbon monoxide and oxides of nitrogen (Table 4).

EPA also is concerned about other pollutants in ambient air, such as air toxics. We analyzed the relationship between air toxics and segregation in a paper published in

2002 (Lopez 2002). Unlike the criteria air pollutants, air toxics, also referred to as hazardous air pollutants (HAPs), do not have set regulatory limits, nor are these pollutants routinely and systematically monitored. However, through its National Air Toxics Assessment (NATA) database, EPA has modeled annual ambient HAP concentrations based on emissions data and estimates of local land uses and population (US EPA 2005). Census tract average concentrations were estimated for each census tract in the continental United States for 1996. The range of HAPs poses problems in summing them. Three methodologies were used in this analysis: simple summation of all HAPs (total unweighted), summation of the estimated lifetime cancer risks for the metropolitan area average concentration level for each HAP (cancer weighted) and the summation of the ratio of estimated metropolitan area average concentration to a benchmark for non-cancer level based on the reference concentration. The association of HAPs and Black-White segregation was assessed in a similar way to that used for the criteria air pollutants. Again, levels of Black-White segregation were associated with higher levels of total HAPs, cancer and non-cancer risks after controlling for other potential metropolitan level explanatory variables.

In addition, the 1996 census tract level concentration estimates allowed for a more detailed study of HAPs and segregation. This study used a Net Difference Score methodology, which describes the probability that a randomly selected Black person within a metro area lives in a census tract with higher levels of HAPs than a randomly selected White person minus the probability that the Black person is living in a census tract with lower levels of HAPs than the White person. In almost every metropolitan area

(out of 331 total) Blacks were more likely to be living in census tracts with higher concentrations of HAPs no matter which cumulative summation methodology was used. The mean Black - White net difference score was 34 for unweighted, 32 for cancer weighted, 31 for non-cancer weighted. In addition, the degree of this inequality was associated with levels of segregation, even after controlling for other potential explanatory factors. Data are available to perform a similar set of analyses for Hispanics and Asians. Again, in almost every metropolitan area, Asians and Hispanics were more likely to be living in census tracts with higher modeled HAPs concentrations, regardless of summation methodology (Table 5). The exceptions tended to be metropolitan areas with small numbers of the subject group. The degree of inequality was also associated with the severity of segregation. This relationship has been observed for earlier years as well (Lopez 2002).

In an expanded analysis of the 1996 NATA data, multivariate modeling examined how segregation patterns across over 300 MSAs mediated potential racial disparities in cancer risk burdens associated with ambient air toxics concentrations (Morello-Frosch and Jesdale 2005). In this study, the generalized index of dissimilarity was used to capture concurrent segregation across multiple racial/ethnic groups (Sakoda 1981; Iceland 2004). Other covariates in this analysis included: state grouping consisting of six broad geographic classifications of the continental United States in order to take into account the regional variation in both the level of racial/ethnic segregation and its historical causes; population density; MSA population size; local area deprivation, as

measured by the Townsend index (Krieger, Chen et al. 2003) and poverty level; and county-level voter turnout as a proxy for community civic engagement.

Figure 2 shows the racial distribution of lifetime estimated cancer risk burdens associated with ambient HAP exposures across three categories of segregation as measured by the generalized dissimilarity index. A descriptive statistic known as the population risk index (PRI) was used to assess potential environmental inequities across segregation, poverty level and racial/ethnic categories. The PRI is a population-weighted average of the census-tract level total estimated cancer risk associated with modeled ambient air toxics exposures (Morello-Frosch, Pastor et al. 2001). The population risk indices for different demographic groups can be compared with each other to graphically assess the extent to which environmental inequities may be occurring. The y-axis on the graph shows a population-weighted individual excess cancer risk estimate for each racial and segregation category. As indicated in the figure legend, each line in the graph represents one of the five racial/ethnic groups. The graph shows two patterns: first, it indicates that cancer risks across all metropolitan areas increase with increasing segregation levels for all racial/ethnic groups and that overall, Hispanics, Asians, followed by African Americans, have some of the highest estimated cancer risk burdens associated with ambient air toxics in metro areas with higher segregation compared to the average across all groups and compared to Whites and Native Americans. Figure 3 shows the racial breakdown of cancer risk burden across poverty levels. Although there is a persistent racial gap across all levels of poverty, there does not appear to be a gradient that increases with rising area-level poverty, which suggests that the effect of

segregation may be independent of the impact of poverty on the exposure burdens across racial categories. This suggests that although segregation concentrates poverty, area-level income and poverty appear to function independently of segregation to impact community health.

To examine these variables in a multivariate analysis, we assessed the relationship between segregation and estimated cancer risk by stratifying by race/ethnicity, and calculating risk ratios for each level of segregation, using low segregation as the referent group. The model controlled for metro area regional grouping, metro area population size, tract-level poverty and material deprivation (Townsend Index) and tract-level population density. Results indicate that increasing segregation amplifies the cancer risks associated with ambient air toxics for all racial groups, although the effect appears to be strongest for Latinos and African Americans (Table 6).

Taken together, these air pollution studies imply that cities with higher levels of segregation have worse air quality. In addition, increased segregation may also be associated with increased racial inequality to exposure and estimated health risk burdens. Increased levels of sulfur dioxide and ozone have been implicated with increased risk of mortality, pulmonary and cardiovascular effects. Carbon monoxide is associated with increased asthma hospitalizations among children.(Neidell 2004; Peel, Tolbert et al. 2005) The health consequences of increased levels of HAPs are less well understood but they were selected for further study by the EPA because of the seriousness of their potential health effects, the relatively high levels of exposures found in ambient air, and

their ubiquity in the environment. Concern has emphasized carcinogenicity, mutagenicity, teratogenicity, and respiratory effects (Leifkauf 2002). Therefore, to the extent that these HAPs may be related to increased risk for cancers and non-cancer health effects, the overall increase in HAP levels associated with segregation may be important. The association between segregation and inequality of exposure is also important. It may imply that segregation contributes to some of the observed racial disparities in health.

Applying Segregation Measures to Study Other Environmental Hazards and Health Outcomes

It remains unclear what it is about socioeconomic inequality and segregation that degrades the health status of those living in hazardous physical and social environments and ultimately leads to environmental health disparities. It is hypothesized that that SES measures, such as segregation, may mediate and compound the adverse effects of hazardous environmental exposures, although this issue has not been thoroughly researched (Evans and Kranowitz 2002). The techniques used to examine relationships between segregation and inequities in ambient air pollution exposures can be applied to other environmental health issues to elucidate socioeconomic drivers of environmental health disparities. Moreover, although the focus of this paper is on residential segregation, links between segregation and environmental health disparities can also be examined in other contexts, such as the workplace (e.g. to examine occupational health disparities) and in schools (e.g. to examine disparities in children's environmental health).

Lead & Residential Pesticide Use

Childhood lead exposure is an environmental hazard for which there have been persistent disparities by race and income. While the prohibition of lead in gasoline and paint has resulted in a decreased risk of lead poisoning for most Americans, there is a continued problem of elevated lead levels for children living in older, substandard housing (Haley and Talbot 2004). As paint surfaces deteriorate, lead dust enters the environment, and toddlers can ingest this lead during normal hand to mouth activities. In addition, as exterior painted surfaces decay, lead can end up in the soils around housing. Children can ingest this lead while playing outside or through the consumption of food grown in these soils (Mielke and Anderson 1983). Increased levels of lead have been associated with an increased risk of a range of cognitive and behavioral outcomes (Needleman, Gunnoe et al. 1979; Needleman 2004).

Interestingly, despite the fact that substandard housing is a major source of lead exposure, no studies appear to have examined this issue in terms of residential segregation. Yet the persistent racial and class-based disparities in childhood lead poisoning suggest that residential segregation may be concentrating communities of color, particularly African Americans, into the poor inner-city neighborhoods with housing that has lead paint and lead contaminated soils (Roberts, Julsey et al. 2003; Breysse, Farr et al. 2004). There are large disparities in elevated blood level rates between Whites and Blacks, with Blacks being 13.5 times as likely to have blood lead

levels above 20 micrograms per deciliter as Whites (Bernard and McGreehin 2003). The role of segregation in causing these disparities, through increased likelihood of exposure to lead contaminated environments, could be investigated further as a way to understand some of the underlying social drivers that make the racial disparities in childhood lead poisoning persist.

Similarly, residential pesticide use is widespread in the United States, with approximately 80-90% of American households using pesticides (Whitmore, Immerman et al. 1994; Landrigan, Claudio et al. 1999). Recent studies indicate that residential exposures to pesticides are associated with adverse birth outcomes (Eskenazi, Bradman et al. 1999; Whyatt, Camann et al. 2002; Perera, Rauh et al. 2003). Although little is known about residential pesticide use among minority populations in the United States, surveys suggest that frequency of use is more intense in public housing and in areas of high population density in housing (Surgan, Congdon et al. 2002). Applying segregation measures to understand patterns of racial and class-based disparities in exposures to urban pesticides could also elucidate how consumer pesticides used to control pests in substandard housing or public housing projects may disproportionately affect certain minority groups.

Stationary and Mobile Pollution Sources

Several environmental justice studies have examined racial disparities associated with environmental hazard burdens although none of these studies have looked

specifically at the question of residential segregation. Similarly, recent research suggests that on average, mobile sources of pollution account for a significant portion of health risks associated with certain pollutants particularly air toxics (Morello-Frosch, Pastor et al. 2001; Reynolds, Von Behren et al. 2002) and that exposure burdens to these sources are inequitably distributed across race and class lines (Gunier, Hertz et al. 2003). The relationship between neighborhood racial make-up and the siting of hazardous facilities has been long researched (U.S. GAO 1983; United Church of Christ 1987; Institute of Medicine 1999; Sadd, Pastor et al. 1999; Pastor, Sadd et al. 2001). In general, these studies have found that both race and income are important predictors of disparate siting, although some have found that income is more important than race and vice versa (Perlin, Setzer et al. 1995; Szasz and Meuser 1997; Fullilove 2004; Pastor, Sadd et al. 2004). The relationship between race and income is complex, given that Black, Hispanic and Asian incomes are significantly lower than White incomes (Sterling, Rosenbaum et al. 1993). There are other potential exposures, perhaps exacerbated by segregation, that warrant further research. Multiple studies have documented these associations, but the next steps, linking these exposures to health outcomes have been less well studied.

Segregation in Relation to Health Outcomes That May be Environmentally Mediated

There are profound racial differences in residential patterns and in environmental exposure burdens. Together, these may imply that segregation and the resulting inequality in the toxicity of residential environments may be contributing to racial

differences in morbidity and mortality. The following suggest some of the potential associations and causal pathways between segregation and health outcomes that are environmentally mediated or that may enhance community vulnerability to the toxic impacts of contaminant exposures.

Adult Mortality

There is a growing body of evidence linking racial segregation to increased mortality risk among both Blacks and Whites, though the risk tends to be greater for Blacks (Polednak 1991; Polednak 1996; Polednak 1997; Collins and Williams 1999; Cooper, Ryley et al. 2001; Cooper, Kennely et al. 2001; Williams and Collins 2001). Overall metropolitan levels of segregation were associated with increased total mortality and avoidable mortality (LaVeist 2003). Controlling for individual risk factors, neighborhoods with high concentrations of Blacks (which would reflect high P* values) have also been found to have higher levels of mortality (Schultz, Williams et al. 2002). The potential causes of these relationships are not well known, but most likely mean increased exposure to social, economic and environmental risk factors (Howard, Andeson et al. 2000; Bosma, van de Mheen et al. 2001; Deaton and Lubotsky 2001; McLaughlin and Stokes 2002). In addition, the quality of health care and other services available to Blacks is lower (Sheifer, Escarse et al. 2000; Leiyu and Starfield 2001). In the context of segregation, they have the potential to act synergistically to raise allostatic levels of stress and simultaneously increase sensitivity to exposures, reduce the ability to access treatment and assistance and reduce the ability to recover from environmentally mediated

illnesses (Wallace 1988; Wallace and Wallace 1998; Massey 2004). Over time, mortality may increase (Fiscella and Franks 1997; Kennedy, Kawachi et al. 1999). Further research on the health effects of segregation and adult mortality might include a better exploration of the health effects of individual pollutants, the study of how pollutants might be working synergistically to increase adverse health outcomes and modeling of the impacts of exposures to pollutants in individuals with overstressed immune systems or who may be disproportionately vulnerable to the effects of pollution exposure due to both area and individual-level factors.

Infant Mortality and Other Birth Outcomes

Since the first studies exploring the relationship between residential segregation and birth outcomes in the United States were published in the 1950's, the literature has been rather limited in scope and volume. It has focused almost exclusively on Black-White disparities in infant mortality rates, and has used a single dimension of segregation at a time, usually a measure of unevenness such as the index of dissimilarity. The research that does exist, however, has addressed the link between segregation and infant mortality from a few angles and at different levels of aggregation, from intra-city explorations of infant mortality rates by neighborhood (Yankauer 1950; Yankauer and Allaway 1958; Yankauer 1990) to inter-city examinations of the variation in black-white infant mortality ratios (Jobu 1972; LaVeist 1993). The literature over the past 50 years has established clear links between residential segregation, infant mortality, and Black-White infant mortality disparities. It is evident that racial inequalities in social

environments engendered by racial segregation have put Black populations at a serious disadvantage relative to White populations, and have had a resounding impact on infant mortality rates among Blacks in the United States (LaVeist 1989; Yankauer 1990; LaVeist 1993; Guest, Almgren et al. 1998). These effects have consistently been shown to be at least partially independent of potential confounders, such as poverty levels (Bird 1995). There are, however, a few serious gaps in the literature to date. First, the literature focuses solely on infant mortality, and does not focus much on links between segregation and other birth outcomes, such as low birth weight. Second, the literature only examines differences between Black and White infant mortality rates, and defines residential segregation as a Black-White phenomenon. Finally, no research has specifically examined the extent to which differential air pollution exposure may mediate and partially explain the relationships between broad social inequalities, neighborhood environments, and persistent racial disparities in birth outcomes.

Analyzing links between segregation, differential exposure to pollution, and birth outcomes among various racial and ethnic groups in the United States would be an important contribution to the literature. More specifically, differences in exposure to air pollutants due to residential segregation may be viewed as the physical manifestations of poor neighborhood environments that lead to poor birth outcomes. Preliminary research indicates that disadvantaged populations often experience a disproportionate amount of air pollution exposure (Woodruff, Parker et al. 2003). Other studies have linked air pollution exposure to negative birth outcomes (Dejmek, Selevan et al. 1999; Ritz and Yu 1999; Dolk, Pattenden et al. 2000; Ritz, Yu et al. 2000; Ritz, Yu et al. 2002) and found

racial disparities in exposure burdens and in relationship to birth outcomes (Ritz and Yu 1999; Ritz, Yu et al. 2000; Ritz, Yu et al. 2002; Woodruff, Parker et al. 2003). Again, none of these studies have examined contextual-level variables in conjunction with individual-level variables that may mediate exposure-outcome relationships. Moreover, these studies have not assessed whether residential segregation amplifies observed associations between adverse birth outcomes and pollution exposures and how these dynamics play out across racial and ethnic groups. Examining this question, particularly in relation to health outcomes that may be environmentally mediated (such as coronary heart disease, mortality, and low birth weight) might help to elucidate how segregation contributes to environmental health disparities.

Asthma

Several factors related to the etiology of asthma may be associated with or exacerbated by segregation. Asthma is often triggered by roaches, dust mites and mold, all of which are linked to housing quality (Platts-Mills, Sporik et al. 1995). Segregation, by limiting Black housing options, may lead to increased exposure to these triggers. Ozone, carbon monoxide, PM₁₀ and other pollutants have been implicated as asthma exacerbators (Leifkauf 2002; Loh and Sugarman-Brozan 2002; Peden 2002). If segregation is linked to increased levels of these pollutants, this may represent another pathway to ill health. Asthma rates are higher among Blacks than Whites (Grant, Lyttle et al. 2000); (CDC 2004). It has been identified as the primary preventable cause of hospitalizations (Flores, Abreu et al. 2003; Masoli, Fabian et al. 2004; Pendergraft,

Stanford et al. 2004) and it is an important cause of mortality (Masoli, Fabian et al. 2004). The economic burden of asthma is in the billions of dollars and the cost of lost school days and work days is similarly high (Gendo and Lodewick 2005). The disparate risk of asthma is heightened by the dearth of access to health care in many Black majority communities. In addition to being less likely to have health insurance, Black majority neighborhoods have been more likely to have their hospitals shut down than other neighborhoods (Sager 1983). In the future, all the components of the causal path leading to poor asthma outcomes might be examined in the context of segregation including, attending schools in segregated districts with disparities in the quality of school facilities, the risk of living in poor quality housing, the relationship between poor housing and exposure to air pollution, the distribution of preventive and emergency care facilities and the role of segregation in access to medical care.

Conclusions and Implications for Policy

Advocates working on environmental justice issues have urged scientists, policymakers, and the regulatory community to consider the junctures of socio-economic inequality, environmental protection, and public health. Certain disparities in exposures to environmental hazards may be related to or mediated by the degree of racial residential segregation, and these exposures may have important clinical and environmental health significance for populations across racial and class lines. Additional research, incorporating new models of exposure and better reflecting the patterns of environmental exposure, should include segregation as a health risk factor. Moreover, while most research has focused on the health consequences of Black-White segregation in

metropolitan areas, other minority groups may be similarly affected. Finally, the health impacts of rural segregation, which were not addressed in this paper, should also be examined.

Although the literature on segregation and health has expanded significantly in recent years, studies that specifically address environmental health disparities are in their infancy. In general, most of this work has been limited to cross-sectional studies. Future research will require the development of longitudinal studies that look simultaneously at people and places—that is the trajectories of individuals in conjunction with the trajectories and evolution of the neighborhoods and metro areas where they live. These studies could also examine residential segregation in conjunction with segregation in other domains such as the educational system and the workplace.

A regional equity perspective is critical to understanding individual and contextual level relationships between racial/ethnic segregation and environmental health disparities (Morello-Frosch 2002b). Racial segregation and other SES disparities play out in major metropolitan areas along divides between the city core and the suburbs and across diverse neighborhoods (Gee 2002; Subramanian, Acevedo-Garcia et al. 2005). Moreover, segmentation of housing markets, the spatial mismatch of labor markets, and the decentralization of metropolitan governance contributes to unequal access to economic opportunities and the fragmentation of local control over land use and zoning decisions in ways that affect community environmental health (Kain 1992; Oliver and Shapiro 1995; Alshutler, Morrill et al. 1999; Conley 1999; Preston and McLafferty 1999;

Keister 2000). As discussed earlier, there is mounting evidence that various aspects of social inequality have contributed to the greater burden of environmental hazard exposure and health risks for communities of color and the poor. Social inequality, such as residential segregation, may affect the options that communities have to address environmental and health problems through, for example, the effects of poverty on the likelihood of having health insurance, or the impact of language limitations on effective engagement with public officials. Therefore, it is necessary to incorporate these broad but significant indicators of community socioeconomic vulnerability and other individual-level factors into a comprehensive understanding of patterns of environmental health disparities. Ultimately this enables policy makers and regulators to understand not only whether a community may be overburdened, but also whether it has the capacity and resources to recover and seek remedies.

How the regulatory community should address fundamental socioeconomic drivers of environmental health remains an open question. The capacity of environmental and public health agencies to proactively address these issues is constrained by legislative mandates that structure the priorities of their research, regulatory, and enforcement activities. Agencies that conduct research can begin to grapple with how to integrate contextual level measures, such as segregation and other measures of SES, with the individual-level factors that have traditionally commanded regulatory attention. Furthermore, research can begin to track the effects of segregation more systematically to assess whether and how segregation contributes to racial disparities in environmental health and to determine the independent effects of

segregation on individual health, and whose health may be most negatively impacted. Indeed, segregation may disparately affect certain racial/ethnic groups more than others, or it is possible that segregation adversely affects the health of all racial groups, even in areas where disparities might persist. By developing indicators of social inequality and segregation and integrating these with environmental health data, regulatory agencies can generate the information necessary for informing regional authorities and community stakeholders about how to address some of the possible drivers of environmental health disparities, whether these relate to the built environment, transportation policies, fair housing, or land use planning. Although environmental and public health agencies may not be able to participate directly in these debates, they can generate the data and scientific information necessary to inform the discussion.

For example, suppose research indicates that segregation appears to amplify observed relationships between poor air quality and certain adverse health outcomes, and that segregation has worse health consequences for members of racial minority groups. Regulatory strategies such as air quality monitoring could be enhanced in segregated neighborhoods where poor air quality is a particular concern. Similarly, this information could help communities and local agencies understand how to target their efforts to reduce emissions from major sources. These targeted monitoring and emission source reduction strategies would likely have to be done in partnership with communities who would play a critical role in helping to identify smaller emissions sources that typically fall below the regulatory radar screen but that may be located near sensitive receptors (e.g. residential communities or schools). Communities can also help agencies grapple

with balancing the need to promote more effective regulation while also promoting economic opportunities within a region. Previous agency-community collaborations of this sort include monitoring and source reduction efforts conducted by the California Air Resources Board and the communities of Barrio Logan in San Diego California and Wilmington in the Los Angeles area.

Rising interest within the regulatory community and the public about the effects of SES and racial inequality on distributions of environmental hazards and their connection to health outcomes necessitates developing new analytical approaches that leverage existing datasets to sort through complex equity issues. Examining these issues through the lens of segregation can reveal connections between individual and contextual factors that shape environmental health disparities, elucidate innovative methodologies aimed at examining environmental justice concerns, and assess the viability of regional approaches to address racial equity issues in pollution control and prevention.

Figures and Tables

Figure 1: Framework for Understanding Segregation and Environmental Health

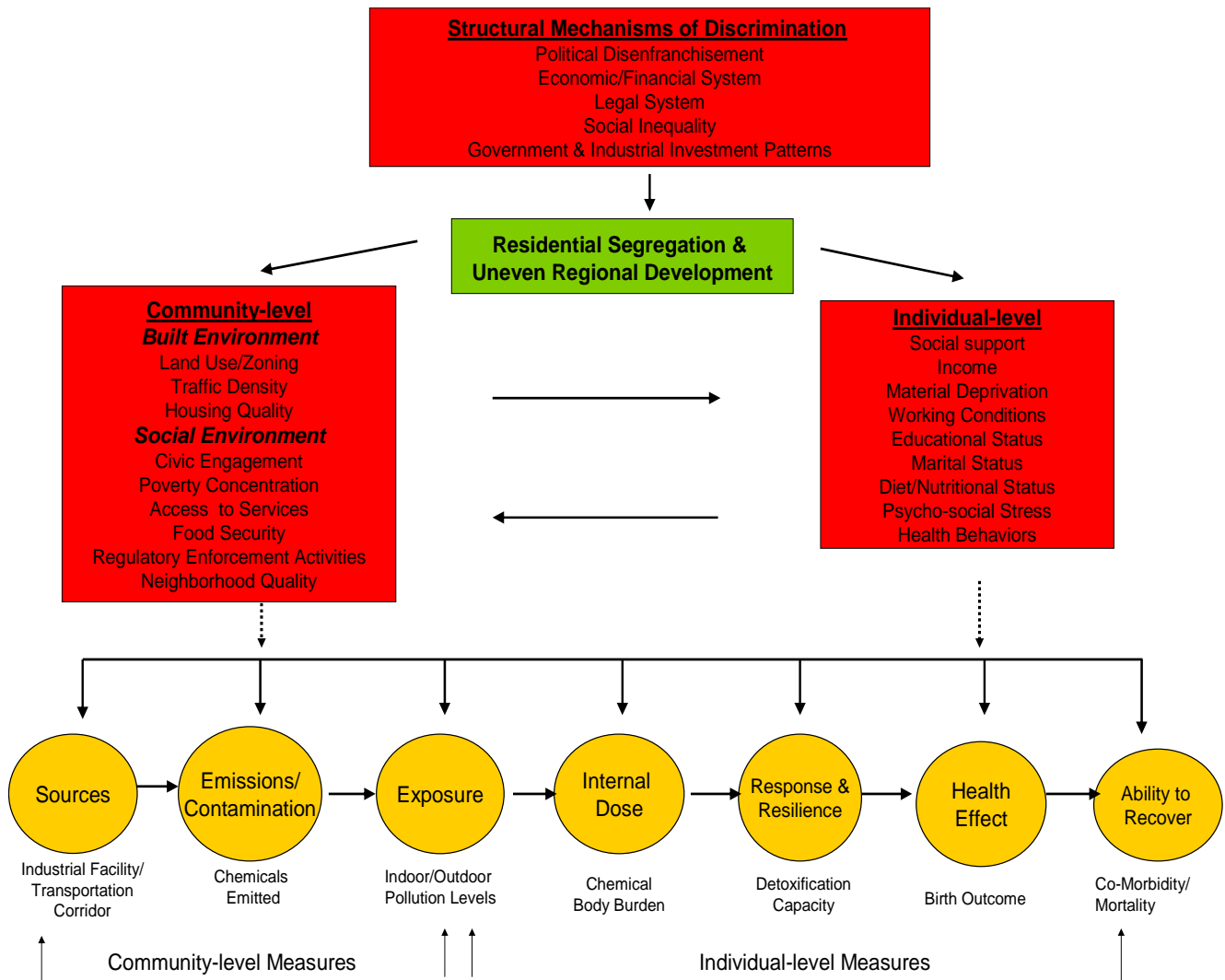


Figure 2

estimated cancer risk by race/ethnicity and racial/ethnic residential segregation, among residents of continental U. S. metropolitan areas

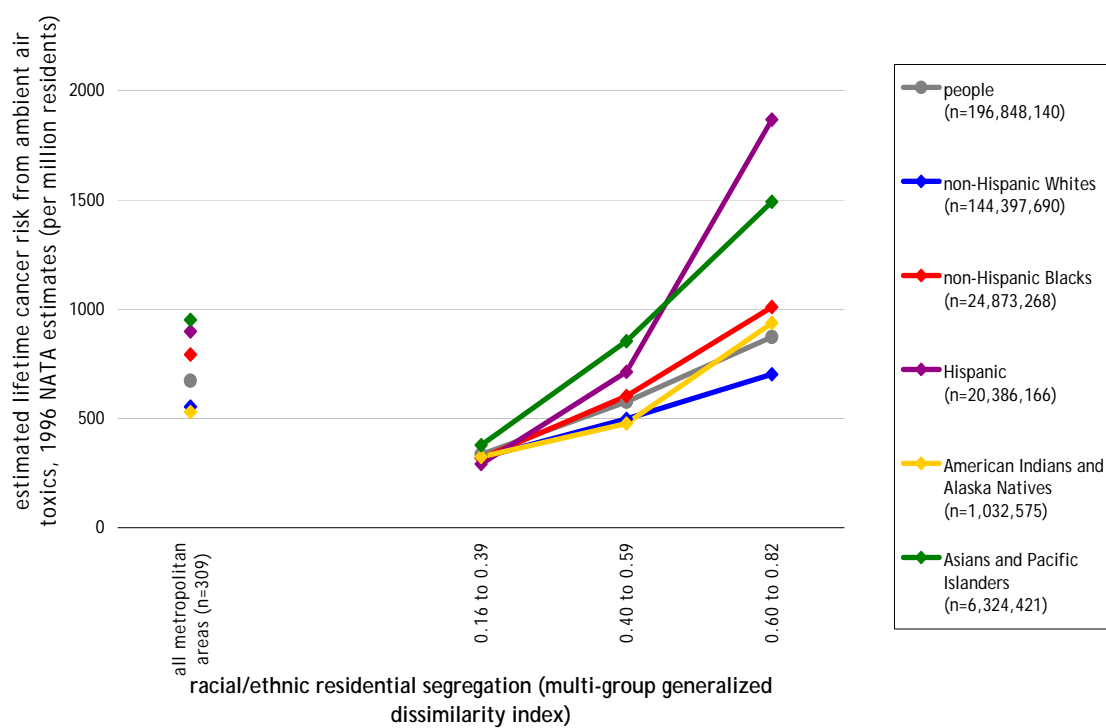


Figure 3

estimated cancer risk by race/ethnicity and poverty status, among
residents of continental U. S. metropolitan areas

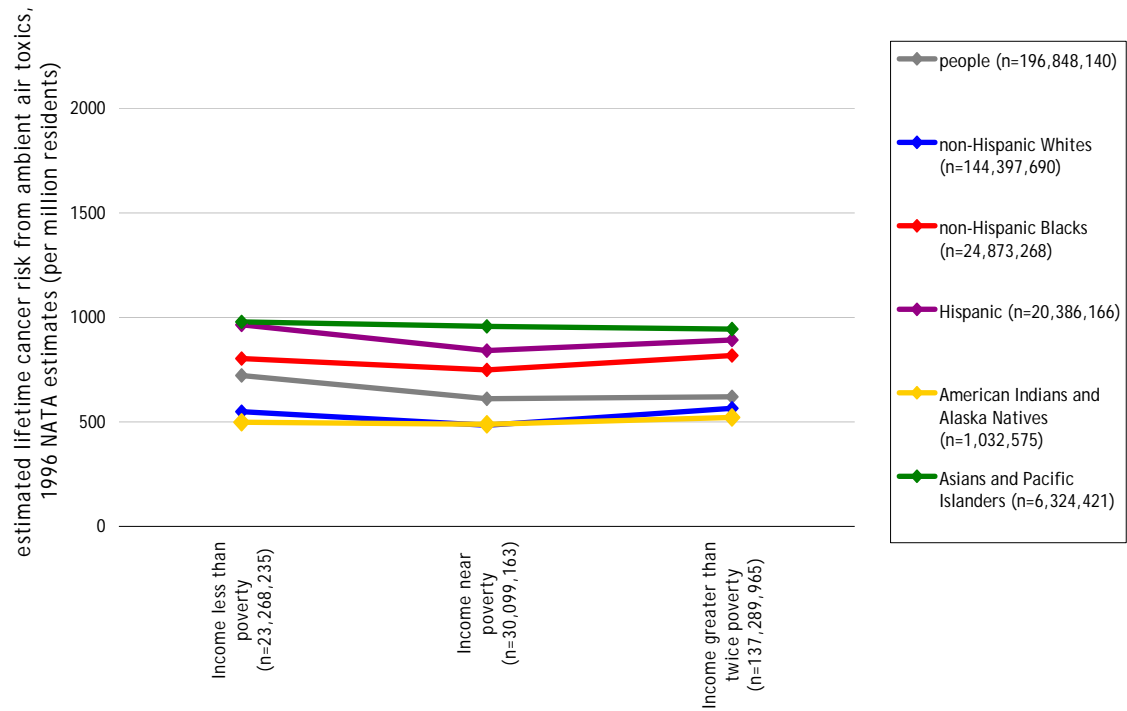


Table 1: Summary Table of Measures for Five Segregation Dimensions

| Measure | dimension | Formula | composition invariant | Multi- group extension | spatial |
|-------------------------------|----------------|--|--------------------------|------------------------------|---------|
| Index of dissimilarity | Evenness | $D = \sum [t_i p_{im} - P_m] / (2T P_m (1 - P_m))$ | yes | yes | no |
| Interaction index | Exposure | ${}_m P_n^* = \sum [(t_i p_{im} / TP_m)(p_{in})]$ | no | no | no |
| Duncan's delta index | Concentration | $DEL = \sum [(t_i p_{im} / TP_m) - (a_i / A)] / 2$ | no | no | no |
| Absolute centralization index | Centralization | $ACE = \sum [X_{i-1p} A_i] - \sum [X_{ip} A_{i-1}]$ tracts sorted by land area $X_{ip} = \sum [t_i p_{im}]$, tracts from 0 to i $A_{ip} = \sum [a_i]$, tracts from 0 to i | no | no | yes |
| Spatial proximity index | Clustering | $SP = (TP_m P_{mm} + TP_n P_{nn}) / NP_{tt}$ $P_{mm} = \sum \sum [(t_i p_{im} t_j p_{jn} c_{ij}) / TP_m TP_n]$ $c_{ij} = e^{-d_{ij}}$ d_{ij} = distance between tract i and tract j . | no | no | yes |

T = number of metro area residents

t_i = number of residents in tract i .

P_m = proportion of metro area residents of racial/ethnic group m .

p_{im} = proportion of tract i 's residents of racial/ethnic group m .

A = land area of metro area

a_i = land area of tract i .

Table 2: Metropolitan Segregation with Whites Dissimilarity Index* for 1980-2000

| | <u>1980</u> | <u>1990</u> | <u>2000</u> |
|---------------------------|-------------|-------------|-------------|
| African Americans | 73.8 | 68.8 | 65.0 |
| American Indians | 37.3 | 36.8 | 33.3 |
| Asian & Pacific Islanders | 41.2 | 42.0 | 42.1 |
| Hispanic | 50.7 | 50.6 | 51.5 |

Source: U.S. Census

**See text for explanation of the dissimilarity index.*

**Table 3: Distribution of Racial/Ethnic Groups by Level of Metropolitan Area Segregation (Generalized Index of Dissimilarity)
Census 2000**

| | Total | low & moderate 0.16 to 0.39 | high 0.40 to 0.59 | extreme 0.60 to 0.82 |
|--|-------------|--------------------------------|----------------------|-------------------------|
| metropolitan areas | 309 | 25% | 53% | 21% |
| census tracts | 45,710 | 10% | 50% | 40% |
| National | 196,848,140 | 11% | 52% | 37% |
| Hispanics of all races | 20,386,166 | 13% | 66% | 21% |
| non-Hispanic whites | 144,397,690 | 12% | 51% | 37% |
| non-Hispanic blacks | 24,873,268 | 5% | 45% | 50% |
| non-Hispanic American Indians & Alaska Natives | 894,954 | 21% | 60% | 19% |
| non-Hispanic Asians & Pacific Islanders | 6,069,605 | 12% | 64% | 24% |
| West Coast | 34,819,823 | 33% | 67% | - |
| Hispanics of all races | 7,756,347 | 20% | 80% | - |
| non-Hispanic whites | 21,565,910 | 42% | 58% | - |
| non-Hispanic blacks | 2,256,761 | 21% | 79% | - |
| non-Hispanic American Indians & Alaska Natives | 233,259 | 50% | 50% | - |
| non-Hispanic Asians & Pacific Islanders | 2,947,432 | 18% | 82% | - |
| South | 39,028,191 | 5% | 71% | 24% |
| Hispanics of all races | 1,983,575 | 2% | 89% | 9% |
| non-Hispanic whites | 28,404,970 | 5% | 72% | 23% |
| non-Hispanic blacks | 7,995,229 | 5% | 63% | 32% |
| non-Hispanic American Indians & Alaska Natives | 110,127 | 10% | 72% | 18% |
| non-Hispanic Asians & Pacific Islanders | 514,659 | 5% | 74% | 20% |
| Mountains & Plains | 10,125,466 | 44% | 45% | 11% |
| Hispanics of all races | 685,376 | 51% | 43% | 5% |
| non-Hispanic whites | 8,507,657 | 44% | 44% | 12% |
| non-Hispanic blacks | 565,269 | 26% | 54% | 19% |
| non-Hispanic American Indians & Alaska Natives | 174,238 | 26% | 71% | 3% |
| non-Hispanic Asians & Pacific Islanders | 184,341 | 52% | 40% | 8% |
| Border | 18,113,094 | 9% | 89% | 2% |
| Hispanics of all races | 4,620,933 | 14% | 85% | 0% |
| non-Hispanic whites | 11,126,767 | 7% | 91% | 2% |
| non-Hispanic blacks | 1,853,246 | 5% | 90% | 5% |
| non-Hispanic American Indians & Alaska Natives | 135,802 | 4% | 95% | 1% |
| non-Hispanic Asians & Pacific Islanders | 351,491 | 4% | 94% | 2% |
| Mid-West | 43,620,713 | 3% | 26% | 72% |
| Hispanics of all races | 1,475,572 | 1% | 12% | 87% |
| non-Hispanic whites | 35,856,980 | 3% | 29% | 68% |
| non-Hispanic blacks | 5,463,371 | 1% | 10% | 90% |
| non-Hispanic American Indians & Alaska Natives | 138,166 | 4% | 41% | 55% |
| non-Hispanic Asians & Pacific Islanders | 656,826 | 3% | 25% | 72% |
| Northeast | 51,140,853 | 1% | 40% | 59% |
| Hispanics of all races | 3,864,361 | 0% | 29% | 70% |
| non-Hispanic whites | 38,935,406 | 2% | 43% | 56% |
| non-Hispanic blacks | 6,739,392 | 0% | 29% | 71% |
| non-Hispanic American Indians & Alaska Natives | 103,362 | 3% | 35% | 63% |
| non-Hispanic Asians & Pacific Islanders | 1,414,856 | 0% | 38% | 61% |

Table 4 Air Pollution and Black White Residential Segregation

| Pollutant | Number of Metropolitan Areas | Coefficient | 95% Confidence Interval |
|--|---|--------------------|--------------------------------|
| Carbon Monoxide | 130 | -0.019 | (-.0021, -.036)* |
| Particulate Matter | 201 | 0.006 | (-.054, .066) |
| Oxides of Nitrogen | 94 | -0.00002101 | (-.0000093, .000051) |
| Sulfur Dioxide | 135 | 0.00004713 | (.000014, .000080)** |
| Ozone | 197 | 0.000233 | (.000097, .00037)** |
| Hazardous Air Pollutants - Total Unweighted | 315 | 0.054 | (.0152, .0928)** |
| Hazardous Air Pollutants - Cancer Weighted | 315 | 1.00663 | (.561, 1.452)** |
| Hazardous Air Pollutants - Non- Cancer Weighted | 315 | 0.014 | (-.0092, .037) |

* Significant at the .05 level

** Significant at the .01 level

Multivariate regression comparing metropolitan area average pollutant level with Black-White dissimilarity index
Regression controlled for metropolitan level percent of people living in poverty, total
population, per capita income, percent of civilian labor force employed in manufacturing

Table 5: Relationship between segregation and inequality of exposure to air toxics

| | Total Unweighted | Cancer Weighted | Non-Cancer Weighted |
|---------------------------------|---------------------------|----------------------------|----------------------------|
| Asian - White Segregation | 0.0034 (-.0018, .0086) | 0.0011 (-.0058, .0080) | 0.0015 (-.0032, .0062) |
| Hispanic - White Segregation | 0.007 (.0029, .0111)** | 0.0059 (.0017, .0192)** | 0.0054 (.0018, .0091)** |
| Black - White Segregation | 0.007 (.0048, .0091)** | 0.0046 (.0021, .0071)** | 0.0052 (.0033, .0092)** |

** Significant at the .01 level

Multivariate regression comparing metropolitan area net difference score with dissimilarity index

Regression controlled for metropolitan level percent of people living in poverty, total population, per capita income, percent of civilian labor force employed in manufacturing and percent of subject group residents.

Table 6: Relative estimated lifetime cancer risk from inhaled pollutants, NATA 1996 census tract-level pollutant estimates, Census 1990 resident characteristics, United States metropolitan areas (n=309)

| | R ² = 38% | | | | | | | | | |
|--|----------------------|---|------|---|-------|----------------------|---|------|---|-------|
| | Highly segregated | | | | | Extremely segregated | | | | |
| total population | 1.04 | (| 1.01 | - | 1.07) | 1.32 | (| 1.28 | - | 1.36) |
| non-Hispanic Whites | 1.04 | (| 1.01 | - | 1.08) | 1.28 | (| 1.24 | - | 1.33) |
| non-Hispanic Blacks | 1.09 | (| 0.98 | - | 1.21) | 1.38 | (| 1.24 | - | 1.53) |
| Hispanics (all Races) | 1.09 | (| 1.01 | - | 1.17) | 1.74 | (| 1.61 | - | 1.88) |
| non-Hispanic American Indians & Alaska Natives | 1.02 | (| 0.77 | - | 1.35) | 1.21 | (| 0.90 | - | 1.64) |
| non-Hispanic Asians & Pacific Islanders | 1.10 | (| 0.97 | - | 1.24) | 1.32 | (| 1.16 | - | 1.51) |

Controlled for census tract-level population density, poverty rate, material deprivation (Townsend index), county-level voter turnout, metropolitan areas size, and U.S. regional state grouping (6 regions).

References

- Acevedo-Garcia, D. K., A. Lochner, et al. (2003). "Future Directions in Residential Segregation and Health Research: A Multilevel Approach." American Journal of Public Health **93**(2): 215-221.
- Alshutler, A., W. Morrill, et al. (1999). Governance and opportunity in metropolitan America. Washington, DC.
- Bernard, S. and M. McGreehin (2003). "Prevalence of blood lead levels ≥ 5 micro g/dL among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood of lead levels 5 to 10 micro g/dL. Third National Health and Nutrition Examination Survey, 1988 - 1994." Pediatrics **112**(6 Pt 1): 308 - 313.
- Bird, S. T. (1995). "Separate Black and White Infant Mortality Models: Differences in the Importance of Structural Variables." Social Science and Medicine **41**(11): 1507-1512.
- Bobo, L. (2001). Racial attitudes and relations at the close of the twentieth century. America Becoming - Racial Trends and their Consequences, Volume I. W. J. W. N. Smelser, and F. Mitchell. Washington, D.C., National Research Council, 2001. **1**: 264-301.
- Bosma, H., D. van de Mheen, et al. (2001). "Neighborhood socioeconomic status and all-cause mortality." American Journal of Epidemiology **153**(4): 363-371.
- Breyse, P., N. Farr, et al. (2004). "The relationship between housing and health: children at risk." Environmental Health Perspectives **112**(15): 1583 - 1588.

- Brunner, E. J. (2000). Toward a new social biology. Social Epidemiology.
- Lisa.F.Berkman and Ichiro.Kawachi. New York, Oxford University Press: 306-331.
- CDC (2004). "Asthma prevalence and control characteristics by race/ethnicity -- United States, 2002." MMWR **53**(7): 145-148.
- Center for Third World Organizing (2002). Roadblocks to Health: Transportation Barriers to Healthy Communities. Oakland, CA, Center for Third World Organizing, People United for a Better Oakland, and Transportation and Land Use Coalition.
- Collins, C. and D. Williams (1999). "Segregation and mortality: The deadly effects of racism." Social Forum **14**: 495-523.
- Conley, D. (1999). Being Black, Living in the Red: Race, Wealth, and Social Policy in America. Berkeley CA, UC Press.
- Cooper, J., T. Ryley, et al. (2001). "Energy use and transport correlation linking personal travel and related uses to urban structure." Environmental Science and Policy **4**(6): 307-316.
- Cooper, R., J. Kennely, et al. (2001). "Relationship between premature mortality and socioeconomic factors in black and white populations of US metropolitan areas." Public Health Reports **116**(5): 464-73.
- Crenshaw, K. (1988). "Race, reform, and retrenchment: transformation and legitimation in antidiscrimination law." Harvard Law Review **101**: 1331-1387.
- Dawkins, C. J. (2004). "Measuring the spatial pattern on residential segregation." Urban Studies **41**(4): 833-851.

- Deaton, A. and D. Lubotsky (2001). Mortality, inequality and race in American cities and states, National Bureau of Economic Research.
- Dejmek, J., S. Selevan, et al. (1999). "Fetal growth and maternal exposure to particulate matter during pregnancy." Environmental Health Perspectives **107**(6): 475-480.
- Diez-Roux, A. (1997). "Neighborhood environments and coronary heart disease: A multilevel analysis." American Journal of Epidemiology **146**: 48-63.
- Diez-Roux, A. (1998). "Bringing context back into epidemiology: Variables and fallacies in multilevel analysis." American Journal of Public Health **88**: 216-222.
- Diez-Roux, A. (2000). "Multilevel analysis in public health research." Annual Review of Public Health **21**: 171-192.
- Dolk, H., S. Pattenden, et al. (2000). "Perinatal and infant mortality and low birth weight among residents near cokeworks and Great Britain." Archives of Environmental Health **55**(1): 26-30.
- Downey, L. (2003). "Spatial measurement, geography, and urban racial inequality." Social Forces **81**(3): 937-952.
- Duncan, O. and B. Duncan (1955a). "The A methodological analysis of segregation indices." American Sociological Review **20**: 210-217.
- Duncan, O. and B. Duncan (1955b). "Residential distribution and occupational stratification." American Journal of Sociology **60**: 493-503.
- EPA. (2004). "Air Data." Retrieved December 13, 2004, 2004, from <http://www.epa.gov/air/data/>.

- Eskenazi, B., A. Bradman, et al. (1999). "Exposures of children to organophosphate pesticides and their potential adverse health effects." Environmental Health Perspectives **107**(Suppl 3): 409-419.
- Evans, G. and E. Kranowitz (2002). "Socioeconomic status and health: The potential role of environmental risk exposure." Annual Review of Public Health **23**: 303-31.
- Farley, J. (1995). "Race Still Matters: The Minimal Role of Income and Housing Cost as Causes of Housing Segregation in St. Louis, 1990." Urban Affairs Review **31**(2): 244-254.
- Farley, R. (1984). "P* segregation indices: what can they tell us about housing segregation in 1980?" Urban Studies **1984**(21): 331-336.
- Fiscella, K. and P. Franks (1997). "Does psychological distress contribute to racial and socioeconomic disparities in mortality?" Social Science and Medicine **45**(12): 1805-1809.
- Flores, G., M. Abreu, et al. (2003). "Keeping children out of hospitals: parents' and physicians' perspectives on how pediatric hospitalizations for ambulatory care-sensitive conditions can be avoided." Pediatrics **112**(5): 1021 - 1030.
- Fullilove, M. (2004). Rootshock: How tearing up city neighborhoods hurts America and what we can do about it. New York, One World Press.
- Gee, G. (2002). "A multi-level analysis of the relationship between institutional and individual racial discrimination and health status." American Journal of Public Health **92**: 615-623.

- Gee, G. and D. Payne-Sturges (2004). "Environmental health disparities: A framework integrating psychosocial and environmental concepts." Environmental Health Perspectives **112**3: 1645–165.
- Gendo, K. and M. Lodewick (2005). "Asthma economics: focusing on therapies that improve costly outcomes." Current Opinions in Pulmonary Medicine **11**(1): 43 - 50.
- Gordon, C. (2003). "Role of environmental stress in the physiological response to chemical toxins." Environmental Research **92**: 1-7.
- Grannis, R. (2002). "Discussion: segregation indices and their functional inputs." Sociological Methodology **32**: 69-84.
- Grant, E., C. Lyttle, et al. (2000). "The relationship of socioeconomic factors and racial/ethnic differences in US asthma mortality." American Journal of Public Health **90**(12): 1923-1925.
- Guest, A. M., G. Almgren, et al. (1998). "The Ecology of Race and Socioeconomic Distress: Infant and Working-Age Mortality in Chicago." Demography **35**(1): 23-34.
- Guhathakurta, S. and M. Wichert (1998). "Who pays for growth in the city of Phoenix: an equity-based perspective on suburbanization." Urban Affairs Review **33**: 813-838.
- Gunier, R., A. Hertz, et al. (2003). "Traffic density in California: socioeconomic and ethnic differences among potentially exposed children." Journal of Exposure Analysis and Environmental Epidemiology **13**(3): 240-246.

- Haley, V. and T. Talbot (2004). "Geographic analysis of blood lead levels in New York state children born 1994 - 1997." Environmental Health Perspectives **112**(15): 577-582.
- Harvey, D. (1989). The Urban Experience. Baltimore, The Johns Hopkins University Press.
- Hersh, R. (1995). Race and Industrial Hazards: An Historical Geography of the Pittsburgh Region, 1900-1990. Washington, DC, Resources for the Future.
- Hise, G. (1997). Magnetic Los Angeles. Baltimore, MD, Johns Hopkins University Press.
- House, J. and D. Williams (2000). Understanding and reducing socioeconomic and racial/ethnic disparities in health. Promoting Health: Intervention Strategies from Social and Behavioral Research. Washington, DC, National Academy Press: 81-124.
- Howard, G., R. Andeson, et al. (2000). "Race, socioeconomic status, and cause-specific mortality." Annals of Epidemiology **10**: 214-223.
- Iceland, J. (2004). "Beyond black and white: Metropolitan residential segregation in multi-ethnic America." Social Science Research **33**: 248-271.
- Iceland, J. and E. Steinmetz (2003). The effects of using census block groups instead of census tracts when examining residential housing patterns, Bureau of the Census.
- Iceland, J., D. Weinberg, et al. (2002). Racial and ethnic segregation in the United States: 1980 - 2000, US Bureau of the Census.
- Institute of Medicine (1999). Toward environmental justice: Research, education, and health policy needs. Washington, DC, Committee on Environmental Justice,

- Health Sciences Policy Program, Health Sciences Section, Institute of Medicine:
14-21.
- James, D. and K. Taeuber (1985). Measures of segregation. Sociological Methodology.
N. Tuma. San Francisco, CA, Jossey-Bass, Inc.: 1-32.
- Jargowsky, P. (1997). Poverty and Place: Ghettos, Barrios, and the American City. New
York, NY, Russell Sage Foundation.
- Jobu, R. M. (1972). "Urban Determinants of Racial Differentiation in Infant Mortality."
Demography **9**(4): 603-615.
- Jones, C. (2001). "'Race,' Racism, and the Practice of Epidemiology." American Journal
of Epidemiology **154**(4): 299-304.
- Kain, J. (1968). "Housing segregation, Negro employment, and metropolitan
decentralization." Quarterly Journal of Economics **82**: 32-59.
- Kain, J. (1992). "The spatial mismatch hypothesis: Three decades later." Housing Policy
Debate **3**(2): 371-460.
- Kaplan, G. and J. Lynch (1999). "Socioeconomic considerations in the Primordial
Prevention of Cardiovascular Disease." Preventive Medicine **26**(6, part 2): S30-5.
- Kawachi, I. and L. Berkman (2003). Neighborhoods and Health. New York, Oxford
University Press.
- Keister, L. (2000). Wealth in America: Trends in Wealth Inequality. New York,
Cambridge University Press.
- Kennedy, B., I. Kawachi, et al. (1999). (Dis)respect and Black mortality. The Society and
Population Reader. I. Kawachi, B. Kennedy and R. Wilkinson. New York, The
New Press.

- Krieger, N. (1994). "Epidemiology and the web of causation: Has anyone seen the spider." Social Science and Medicine **39**(7): 887-903.
- Krieger, N. (1999). "Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination." International Journal of Health Services **29**(2): 295-352.
- Krieger, N., J. Chen, et al. (2003). "Choosing area-based socioeconomic measures to monitor social inequalities in low birthweight and childhood lead poisoning—The Public Health Disparities Geocoding Project (US)." Journal of Epidemiology and Community Health **57**: 186–199.
- Landrigan, P., L. Claudio, et al. (1999). "Pesticides and inner-city children: exposures, risks, and prevention." Environmental Health Perspectives **107**(suppl 3): 431-437.
- LaVeist, T. (1989). "Linking residential segregation to the infant mortality disparity in U.S. cities." Sociology and Social Science Research **73**(2): 90-94.
- LaVeist, T. (2003). "Racial segregation and longevity among African Americans: an individual-level analysis." Health Services Research **38**(6 Pt. 2): 1719 - 1733.
- LaVeist, T. A. (1993). "Segregation, Poverty, and Empowerment: Health Consequences for African Americans." Milbank Quarterly **71**(1): 41-64.
- Lawrence, C. R. (1987). "The id, the ego, and equal protection: reckoning with unconscious racism." Stanford Law Review **39**: 317-387.
- Leifkauf, G. (2002). "Hazardous Air Pollutants and Asthma." Environmental Health Perspectives **110**(supplement 4): 505–526.

- Leiyu, S. and B. Starfield (2001). "The effect of primary care physician supply and income inequality on mortality among Blacks and Whites in US metropolitan areas." American journal of Public Health **91**(8): 1246-1250.
- Link, B. and J. Phelan (1995). "Social conditions as fundamental causes of disease." Journal of Health and Social Behavior **42(extra issue)**: 80-94.
- Logan, J. and H. Molotch (1987). Urban Fortunes. Berkeley, University of California Press.
- Loh, P. and J. Sugarman-Brozan (2002). "Environmental justice organizing for environmental health: Case study on asthma and diesel exhaust in Roxbury, Massachusetts." Annals of AAPSS **584**(1): 110-124.
- Lopez, R. (2002). "Segregation and Black/White Differences in Exposure to Air Toxics in 1990." Environmental Health Perspectives **110**(Supplement 2): 289-295.
- Macintyre, S., A. Ellaway, et al. (2002). "Place effects on health: how can we conceptualise, operationalise and measure them?" Social Science and Medicine **55**: 125-139.
- Masoli, M., D. Fabian, et al. (2004). "The global burden of asthma: executive summary of the GINA Dissemination Committee report." Allergy **59**(5): 469 - 478.
- Massey, D. (2004). "Segregation and stratification." Du Bois Review **1**(1): 7-25.
- Massey, D. and N. Denton (1993). American Apartheid: Segregation and the Making of the Underclass. Cambridge, MA, Harvard University Press.
- Massey, D. and E. Fong (1990). "Segregation and neighborhood quality: Blacks, Hispanics, and Asians in the San Francisco metropolitan area." Social Forces **69**: 15-32.

- Massey, D. and A. Gross (1994). "Migration, Segregation, and the Geographic Concentration of Poverty." American Sociological Review **59**: 425-445.
- Massey, D., A. Gross, et al. (1991). "Segregation, the concentration of poverty, and the life chances of individuals." Social Science Research **20**: 397-420.
- Massey, D., M. White, et al. (1996). "The Dimensions of Segregation Revisited." Sociological Methods and Research **25**(2): 172-206.
- Massey, D. S. and N. A. Denton (1988). "The Dimensions of Residential Segregation." Social Forces **67**(2): 281-315.
- McEwen, B. and E. Lasley (2002). The End of Stress as We Know It. Washington, DC, The John Henry Press.
- McLaughlin, D. and S. Stokes (2002). "Income inequality and mortality in US counties: Does minority racial concentration matter?" American Journal of Public Health **92**(1): 99-104.
- Mielke, H. and J. e. a. Anderson (1983). "Lead concentrations in inner-city soils as a factor in the child lead problem." American Journal of Public Health **73**(12): 1366 - 1369.
- Morello-Frosch, R. (2002b). "The Political Economy of Environmental Discrimination." Environment and Planning C **20**: 477-496.
- Morello-Frosch, R. and B. Jesdale (2005). "Separate and Unequal: Residential Segregation and Air Quality in the Metropolitan U.S." Under Review.
- Morello-Frosch, R., M. Pastor, et al. (2002a). "Environmental Justice and Regional Inequality in Southern California: Implications for Future Research." Environmental Health Perspectives **110**((Supplement 2).): 149-154.

- Morello-Frosch, R., M. Pastor, et al. (2001). "Environmental Justice and Southern California's 'Riskscape': The Distribution of Air Toxics Exposures and Health Risks Among Diverse Communities." Urban Affairs Review **36**(4): 551-578.
- Morello-Frosch RA, P. M., Porras C, Sadd J (2002). "Environmental Justice and Regional Inequality in Southern California: Implications for Future Research." Environmental Health Perspectives **110**((Supplement 2).): 149-154.
- Morland, K., S. Wing, et al. (2002). "Neighborhood characteristics associated with the location of food stores and food service places." American Journal of Preventive Medicine **22**: 23–29.
- Mumford Center (2000). Metropolitan racial and ethnic change - Census 2000, Mumford Center.
- Navarro, V. (2002). The Political Economy of Social Inequalities: Consequences for Health and Quality of Life. New York, Baywood.
- Needleman, H. (2004). "Lead poisoning." Annual Review of Medicine. **55**: 209 - 222.
- Needleman, H., C. Gunnoe, et al. (1979). "Deficits in psychologic and classroom performance of children with elevated dentine lead levels." New England Journal of Medicine **300**: 689 - 695.
- Neidell, M. (2004). "Air pollution, health and socio-economic status: the effect of outdoor air quality on childhood asthma." Journal of Health Economics **23**(6): 1209-36.
- Oliver, M. and T. Shapiro (1995). Black Wealth/White Wealth: A New Perspective on Racial Inequality. New York, Routledge.

- Pastor, M. (2001). "Common Ground at Ground Zero? The New Economy and the New Organizing in Los Angeles." Antipode **33**(2): 260-289.
- Pastor, M., P. Dreier, et al. (2000). Regions That Work: How Cities and Suburbs Can Grow Together., University of Minnesota Press.
- Pastor, M., J. Sadd, et al. (2001). "Which came first? Toxic facilities, minority move-in, and environmental justice." Journal of Urban Affairs **23**(1): 1-21.
- Pastor, M., J. Sadd, et al. (2004). "Waiting to Inhale: The Demographics of Toxic Air Releases in 21st Century California." Social Science Quarterly **85**(2): 420-440.
- Peden, D. (2002). "Pollutants and asthma: role of air toxics." Environmental Health Perspectives **110**(Supplement 4): 565-568.
- Peel, J. L., P. E. Tolbert, et al. (2005). "Ambient air pollution and respiratory emergency department visits." Epidemiology **16**(2): 164-74.
- Peet, R. (1984). "Class Struggle, The Relocation of Employment and Economic Crisis." Science and Society **XLVIII**(1): 38-51.
- Pendergraft, T., R. Stanford, et al. (2004). "Rates and characteristics of intensive care unit admissions and intubations among asthma-related hospitalizations." Annals of Allergy and Asthma Immunology **93**(1): 29 - 35.
- Perera, F., V. Rauh, et al. (2003). "Effects of transplacental exposure to environmental pollutants on birth outcomes in a multi-ethnic population." Environmental Health Perspectives **111**(2): 201-205.
- Perlin, S. A., R. W. Setzer, et al. (1995). "Distribution of industrial air emissions by income and race in the United States: An approach using the Toxic Release Inventory." Environmental Science & Technology **29**(1): 69-80.

- Platts-Mills, T., R. Sporik, et al. (1995). "Is there a dose-response relationship between exposure to indoor allergens and symptoms of asthma?" The Journal of Allergy and Clinical Immunology **96**(4): 435-440.
- Polednak, A. (1991). "Black-White differences in infant mortality in 38 standard metropolitan areas." American Journal of Public Health **81**(11): 1480-1482.
- Polednak, A. (1996). "Segregation, discrimination and mortality in U.S. Blacks." Ethnicity and Disease **6**: 99-107.
- Polednak, A. (1997). Segregation, poverty, and mortality in urban African Americans. New York, Oxford University Press.
- Preston, V. and S. McLafferty (1999). "Spatial mismatch research in the 1990s: progress and potential." Papers in Regional Science **78**: 387-402.
- Pulido, L. (2000). "Rethinking Environmental Racism: White Privilege and Urban Development in Southern California." Annals of the Association of American Geographers **90**(1): 12-40.
- Pulido, L., S. Sidawi, et al. (1996). "An Archeology of Environmental Racism in Los Angeles." Urban Geography **17**(5): 419-439.
- Rauh, V., R. Whyatt, et al. (2004). "Developmental effects of exposure to environmental tobacco smoke and material hardship among inner-city children." Neurotoxicology and Teratology **26**: 373-385.
- Reardon, S. F. and G. Firebaugh (2002). "Measures of multigroup segregation." Sociological Methodology **32**: 33-67.
- Reynolds, P., J. Von Behren, et al. (2002). "Traffic patterns and childhood cancer incidence rates in California, United States." Cancer Causes and Control **13**.

- Rios, R., G. Poje, et al. (1993). "Susceptibility to environmental pollutants among minorities." Toxicology and Industrial Health **9**: 797-820.
- Ritz, B. and F. Yu (1999). "The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993." Environmental Health Perspectives **107**(1): 17-25.
- Ritz, B., F. Yu, et al. (2000). "Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993." Epidemiology **11**(5): 502-11.
- Ritz, B., F. Yu, et al. (2002). "Ambient air pollution and risk of birth defects in Southern California." American Journal of Epidemiology **155**(1): 17-25.
- Roberts, J., T. Julsey, et al. (2003). "Using geographic information systems to assess risk for elevated blood lead levels in children." Public Health Reports **118**(3): 221-229.
- Sadd, J., M. Pastor, et al. (1999). "'Every Breath You Take.' The Demographics of Toxic Air Releases in Southern California." Economic Development Quarterly **13**(2): 107-123.
- Sager, A. (1983). "Why urban voluntary hospitals close." Health Services Research **18**(3): 451-481.
- Sakoda (1981). "A generalized index of dissimilarity." Demography **18**: 245-250.
- Sampson, R. (1987). "Urban black violence: The effect of male joblessness and family disruption." American Journal of Sociology **93**(348-382).
- Schultz, A., D. Williams, et al. (2002). "Racial and Spatial Relations as Fundamental Determinants of Health in Detroit." Milbank Quarterly **80**(4): 677-707.

- Sheifer, S., J. Escarse, et al. (2000). "Race and sex differences in the management of coronary artery disease." American Heart Journal **139**(5): 848 - 857.
- Shenassa, E., A. Stubbendick, et al. (2004). "Social Disparities in Housing and Related Pediatric Injury: A Multilevel Study." American Journal of Public Health **94**(4): 633-639.
- Sinton, P. (1997). Fewer Blacks, Latinos Get Loans. San Francisco Chronicle. San Francisco, CA: D1, D10.
- Stearns and Logan (1986). "Measuring trends in segregation. Three dimensions, three measures." Urban Affairs Quarterly **22**(1): 124-150.
- Sterling, T., W. Rosenbaum, et al. (1993). "Income, race and mortality." Journal of the National Medical Association **85**(12): 906-911.
- Subramanian, S. V., D. Acevedo-Garcia, et al. (2005). "Racial residential segregation and geographic heterogeneity in black/white disparity in poor self-rated health in the U.S.: a multi-level statistical analysis." Social Science and Medicine **60**: 1667-1679.
- Surgan, M., T. Congdon, et al. (2002). Pest control in public housing, schools and parks: urban children at risk. LAW 180-4 PESP 202-7643. Albany, New York, State Department of Law, Environmental Protection Bureau.
- Szasz, A. and M. Meuser (1997). "Environmental Inequalities: Literature Review and Proposals for New Directions in Research and Theory." Current Sociology **45**(3): 99-120.

- U.S. GAO (1983). Siting of hazardous waste landfills and their correlation with racial and economic status of surrounding communities. Gaithersburg, MD, U.S. General Accounting Office.
- United Church of Christ (1987). A national report on the racial and socio-economic characteristics of communities with hazardous waste sites. New York, NY, United Church of Christ, Commission for Racial Justice.
- US EPA. (2005). "The." The National-Scale Air Toxics Assessment (available at: <http://www.epa.gov/ttn/atw/nata/>) Retrieved January 4, 2005, from <http://www.epa.gov/ttn/atw/nata/>.
- Walker, R. (1981). A Theory of Suburbanization. Urbanization and Urban Planning in a Capitalist Society. M. Dear and A. Scott. New York, Methuen: 383-429.
- Walker, R. (1985). Class, Division of Labour and Employment in Space. Social Relations and Spatial Structures. D. Gregory and J. Urry. London, Macmillan Publishers Ltd: 164-189.
- Wallace, D. and R. Wallace (1998). A plague on your houses: How New York was burned down and national public health crumbled. New York City, Verso.
- Wallace, R. (1988). "A synergism of plagues: Planned shrinkage, contagious housing destruction and AIDS in the Bronx." Environmental Research **47**(1): 1-33.
- Wallace, R. (1990). "Urban desertification, public health and public order: "Planned shrinkage," violent death, substance abuse and AIDS in the Bronx." Social Science and Medicine **31**: 801-813.
- Wellman, D. (1993). Portraits of White Racism. New York, Cambridge University Press.

- White, M. (1986). "Segregation and diversity: measures in population distribution." Population Review **52**: 198-221.
- Whitmore, R., F. Immerman, et al. (1994). "Non-occupational exposures to pesticides for residents of two U.S. cities." Archives of Environmental Contamination and Toxicology **26**: 47-59.
- Whyatt, R., D. Camann, et al. (2002). "Residential pesticide use during pregnancy among a cohort of urban minority women." Environmental Health Perspectives **110**: 507-514.
- Williams, D. and C. Collins (2001). "Racial residential segregation: a fundamental cause of racial disparities in health." Public Health Reports **116**: 404-416.
- Wilson, W. (1996). When Work Disappears: The World of the New Urban Poor. New York, Alfred A. Knopf.
- Wong, D. W. (2004). "Comparing traditional and spatial segregation measures: a spatial scale perspective." Urban Geography **25**(1): 66-82.
- Woodruff, T., J. Parker, et al. (2003). "Disparities in exposure to air pollution during pregnancy." Environmental Health Perspectives **11**: 942-946.
- Yankauer, A. (1950). "The Relationship of Fetal and Infant Mortality to Residential Segregation: An Inquiry into Social Epidemiology." American Sociological Review **15**(5): 644-648.
- Yankauer, A. (1990). "What Infant Mortality Tells Us." American Journal of Public Health **80**(6): 653-654.

- Yankauer, A. and N. C. Allaway (1958). "The Relation of Indices of Fetal and Infant Loss to Residential Segregation: A Follow-Up Report." American Sociological Review **23**(5): 573-578.
- Yen, I. and S. Syme (1999). "The social environment and health: a discussion of the epidemiologic literature." Annual Review of Public Health **20**: 287–308.